

WEST Search History

DATE: Thursday, June 23, 2005

| Hide? | Set Name | Query | Hit Count |
|--------------------------|----------|---------------------------------------------------------------------|-----------|
| | | <i>DB=PGPB,USPT,EPAB,JPAB,DWPI; THES=ASSIGNEE; PLUR=YES; OP=ADJ</i> | |
| <input type="checkbox"/> | L7 | stegmann T.in. | 5 |
| | | <i>DB=USPT; THES=ASSIGNEE; PLUR=YES; OP=ADJ</i> | |
| <input type="checkbox"/> | L6 | L5 | 5 |
| | | <i>DB=PGPB,USPT,EPAB,JPAB,DWPI; THES=ASSIGNEE; PLUR=YES; OP=ADJ</i> | |
| <input type="checkbox"/> | L5 | L4 and human | 22 |
| <input type="checkbox"/> | L4 | L3 and coronary | 23 |
| <input type="checkbox"/> | L3 | L2 and FGF | 27 |
| <input type="checkbox"/> | L2 | L1 and ischemic | 136 |
| <input type="checkbox"/> | L1 | revascularizing | 315 |

END OF SEARCH HISTORY

FILE COVERS 1907 - 23 Jun 2005 VOL 142 ISS 26
FILE LAST UPDATED: 22 Jun 2005 (20050622/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate
substance identification.

=> "vascular endothelial growth factor"

142221 "VASCULAR"

4 "VASCULARS"

142224 "VASCULAR"

("VASCULAR" OR "VASCULARS")

93569 "ENDOTHELIAL"

10 "ENDOTHELIALS"

93573 "ENDOTHELIAL"

("ENDOTHELIAL" OR "ENDOTHELIALS")

1197732 "GROWTH"

4197 "GROWTHS"

1199890 "GROWTH"

("GROWTH" OR "GROWTHS")

902352 "FACTOR"

804002 "FACTORS"

1424586 "FACTOR"

("FACTOR" OR "FACTORS")

L1 15357 "VASCULAR ENDOTHELIAL GROWTH FACTOR"

("VASCULAR"(W)"ENDOTHELIAL"(W)"GROWTH"(W)"FACTOR")

=> revasculariz5

L2 5881693 5

=> L1 and l2

L3 3740 L1 AND L2

=> "ischemic" and L3

41674 "ISCHEMIC"

7 "ISCHEMICS"

41676 "ISCHEMIC"

("ISCHEMIC" OR "ISCHEMICS")

L4 221 "ISCHEMIC" AND L3

=> human and L4

1441812 HUMAN

327188 HUMANS

1605232 HUMAN

(HUMAN OR HUMANS)
L5 124 HUMAN AND L4

=> physical (w) glue
410840 PHYSICAL
20 PHYSICALS
410860 PHYSICAL
(PHYSICAL OR PHYSICALS)
464811 PHYS
9 PHYSES
464820 PHYS
(PHYS OR PHYSES)
814088 PHYSICAL
(PHYSICAL OR PHYS)
15341 GLUE
4605 GLUES
17735 GLUE
(GLUE OR GLUES)
L6 1 PHYSICAL (W) GLUE

=> physiological (w) glue
47356 PHYSIOLOGICAL
256155 PHYSIOL
1 PHYSIOLS
256155 PHYSIOL
(PHYSIOL OR PHYSIOLS)
282106 PHYSIOLOGICAL
(PHYSIOLOGICAL OR PHYSIOL)
15341 GLUE
4605 GLUES
17735 GLUE
(GLUE OR GLUES)
L7 2 PHYSIOLOGICAL (W) GLUE

=> L7 and L5
L8 0 L7 AND L5

=> heparin and L5
45329 HEPARIN
1716 HEPARINS
45437 HEPARIN
(HEPARIN OR HEPARINS)
L9 6 HEPARIN AND L5

=> "coronary artery stenosis"
58569 "CORONARY"

225 "CORONARIES"
 58635 "CORONARY"
 ("CORONARY" OR "CORONARIES")
 118088 "ARTERY"
 32019 "ARTERIES"
 129534 "ARTERY"
 ("ARTERY" OR "ARTERIES")
 4910 "STENOSIS"
 L10 374 "CORONARY ARTERY STENOSIS"
 ("CORONARY"(W)"ARTERY"(W)"STENOSIS")

=> L10 and L5
 L11 0 L10 AND L5

=> L10 and L4
 L12 0 L10 AND L4

=> D L9 IBIB ABS 1-6
 => "endothelia cell growth factor"
 1099 "ENDOTHELIA"
 1927276 "CELL"
 1699605 "CELLS"
 2565531 "CELL"
 ("CELL" OR "CELLS")
 1197732 "GROWTH"
 4197 "GROWTHS"
 1199890 "GROWTH"
 ("GROWTH" OR "GROWTHS")
 902352 "FACTOR"
 804002 "FACTORS"
 1424586 "FACTOR"
 ("FACTOR" OR "FACTORS")
 L13 0 "ENDOTHELIA CELL GROWTH FACTOR"
 ("ENDOTHELIA"(W)"CELL"(W)"GROWTH"(W)"FACTOR")

=> "astroglial growth factor"
 2419 "ASTROGLIAL"
 1197732 "GROWTH"
 4197 "GROWTHS"
 1199890 "GROWTH"
 ("GROWTH" OR "GROWTHS")
 902352 "FACTOR"
 804002 "FACTORS"
 1424586 "FACTOR"
 ("FACTOR" OR "FACTORS")
 L14 21 "ASTROGLIAL GROWTH FACTOR"

("ASTROGLIAL"(W)"GROWTH"(W)"FACTOR")

=> L14 and L2

L15 6 L14 AND L2

=> D L15 IBIB ABS 1-6

=> "retinal-derived growth factor"

27702 "RETINAL"

388 "RETINALS"

27735 "RETINAL"

("RETINAL" OR "RETINALS")

755252 "DERIVED"

2 "DERIVEDS"

755254 "DERIVED"

("DERIVED" OR "DERIVEDS")

1197732 "GROWTH"

4197 "GROWTHS"

1199890 "GROWTH"

("GROWTH" OR "GROWTHS")

902352 "FACTOR"

804002 "FACTORS"

1424586 "FACTOR"

("FACTOR" OR "FACTORS")

L16 4 "RETINAL-DERIVED GROWTH FACTOR"

("RETINAL"(W)"DERIVED"(W)"GROWTH"(W)"FACTOR")

=> ischemic and L16

41674 ISCHEMIC

7 ISCHEMICS

41676 ISCHEMIC

(ISCHEMIC OR ISCHEMICS)

L17 0 ISCHEMIC AND L16

=> L1 and ischemic

41674 ISCHEMIC

7 ISCHEMICS

41676 ISCHEMIC

(ISCHEMIC OR ISCHEMICS)

L18 883 L1 AND ISCHEMIC

=> "thoractomy incision"

0 "THORACTOMY"

3218 "INCISION"

834 "INCISIONS"

3797 "INCISION"

L19 ("INCISION" OR "INCISIONS")
0 "THRORACTOMY INCISION"
("THRORACTOMY"(W)"INCISION")

=> coronary (w) artery (w) stenosis

58569 CORONARY
225 CORONARIES
58635 CORONARY
(CORONARY OR CORONARIES)
118088 ARTERY
32019 ARTERIES
129534 ARTERY
(ARTERY OR ARTERIES)
4910 STENOSIS

L20 374 CORONARY (W) ARTERY (W) STENOSIS

=> L20 and L18

L21 0 L20 AND L18

=> L20 and L18

L22 0 L20 AND L18

=> L20 and l1

L23 0 L20 AND L1

=> "heparin binding growth factor=> aFGF and L20

823 AFGF
4 AFGFS
823 AFGF
(AFGF OR AFGFS)
L24 0 AFGF AND L20

=> FGF and L20

11142 FGF
1023 FGFS
11273 FGF
(FGF OR FGFS)
L25 0 FGF AND L20

=> "eye-derived growth factor"

104518 "EYE"
23506 "EYES"
112942 "EYE"
("EYE" OR "EYES")
755252 "DERIVED"
2 "DERIVEDS"

755254 "DERIVED"
("DERIVED" OR "DERIVEDS")
1197732 "GROWTH"
4197 "GROWTHS"
1199890 "GROWTH"
("GROWTH" OR "GROWTHS")
902352 "FACTOR"
804002 "FACTORS"
1424586 "FACTOR"
("FACTOR" OR "FACTORS")
L26 52 "EYE-DERIVED GROWTH FACTOR"
("EYE"(W)"DERIVED"(W)"GROWTH"(W)"FACTOR")

=> L26 and ischemic
41674 ISCHEMIC
7 ISCHEMICS
41676 ISCHEMIC
(ISCHEMIC OR ISCHEMICS)
L27 0 L26 AND ISCHEMIC

=> prostatropin and l2
6 PROSTATROPIN
24 PROSTATROPINS
26 PROSTATROPIN
(PROSTATROPIN OR PROSTATROPINS)
L28 9 PROSTATROPIN AND L2

=> D L28 IBIB ABS 1-9

=> glial maturation factor
22883 GLIAL
6 GLIALS
22885 GLIAL
(GLIAL OR GLIALS)
65951 MATURATION
77 MATURATIONS
65982 MATURATION
(MATURATION OR MATURATIONS)
902352 FACTOR
804002 FACTORS
1424586 FACTOR
("FACTOR" OR "FACTORS")
L29 23 GLIAL MATURATION FACTOR
(GLIAL(W)MATURATION(W)FACTOR)

=> ishemic and l29

L30 2 ISHCCEMIC
0 ISHCCEMIC AND L29

=> FGF

L1 20226 FGF

=> vasculariz4

L2 7099699 4

=> L1 and L2

L3 4515 L1 AND L2

=> ischemic and l3

L4 101 ISCHEMIC AND L3

=> neoangiogenesis

L5 1 NEOANGIOGENESIS

=> human and L3

L6 1745 HUMAN AND L3

=> L6 and L4

L7 43 L6 AND L4

=> acidic and l7

L8 3 ACIDIC AND L7

⇒ L7

⇒

L13 43 L7

=> intramyocardiaS3

L14 0 INTRAMYOCARDIAS3

=> myocardia2

SYSTEM LIMITS EXCEEDED - SEARCH ENDED

The search profile you entered was too complex or gave too many answers. Simplify or subdivide the query and try again. If you have exceeded the answer limit, enter DELETE HISTORY at an arrow prompt (=>) to remove all previous answers sets and begin at L1. Use the SAVE command to store any important profiles or answer sets before using DELETE HISTORY.

=> myocardiactly

L15 0 MYOCARDIACALLY

=> hear

L16 1969 HEAR

=> L13 and L16

L17 0 L13 AND L16

=> coronary and L13

L18 21 CORONARY AND L13

=> D L18 IBIB ABS 1-21

L9 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1998:339214 CAPLUS

DOCUMENT NUMBER: 129:63235

TITLE: Intramyocardial infusion of **FGF-1** mimics
ischemic preconditioning in pig myocardium

AUTHOR(S): Htun, Patrik; Ito, Wulf D.; Hoefer, Imo E.; Schaper,
Jutta; Schaper, Wolfgang

CORPORATE SOURCE: Max-Planck-Inst. Physiol. Clin. Res., Bad Nauheim,
D-61231, Germany

SOURCE: Journal of Molecular and Cellular Cardiology (1998),
30(4), 867-877

CODEN: JMCDAJ; ISSN: 0022-2828

PUBLISHER: Academic Press Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Previous studies on the mRNA and protein level suggested a cardioprotective role of **FGF-1**. These presumed actions of **FGF-1** and **FGF-2**, as well as the underlying mechanisms, were investigated in this study. Human recombinant **FGF-1** (0.5 µg/mL, 20 µl/min) and **FGF-2** (2 µg/mL) were applied by direct intramyocardial infusion (IM) for 60 min prior to a 60 min LSD-occlusion and 120 min reperfusion. Myocardial infarction compared to the region at risk was significantly decreased by **FGF-1** and **FGF-2** treatment (**FGF-1**: 51.8%, **FGF-2**: 57.3% vs. control 83.4%). The increase in survival time was about 33 min. and equaled that of **ischemic** preconditioning. This effect was caused by the mitogenic part of the mol., since infusion of a truncated version of **FGF-1** (0.5-1 µg/mL), lacking mitogenicity but maintaining hemodynamic activity, did not induce cardioprotection (78.3% vs. control 83.4%). Suramin (0.5 µg/mL) prevented the observed cardioprotection (77.0% vs. control 83.4%) proving that the cardioprotective effect is receptor-mediated. Genistein (0.5 µg/mL), an inhibitor of tyrosine kinases, abolished the cardioprotection as well (77.2% vs. control: 83.4%). Immunohistochem. staining revealed an uptake and translocation of exogenous **FGF-1** to a (peri-)nuclear localization in myocytes and into non-myocytes for **FGF-2**. The authors conclude that both **FGF-1** and **FGF-2** are cardioprotective (**FGF-1** being more active on a molar basis), and mimic **ischemic** preconditioning. Their actions are receptor-mediated and receptor activation is involved. Uptake and transport to a (peri-)nuclear localization, seems to be a pathway of minor relevance, since it could not be blocked by tyrosine kinase receptor localization, seems to be a pathway of minor relevance, since it could not be blocked by tyrosine kinase receptor inhibition. Tyrosine kinase-coupled receptor occupation in general is not protective as demonstrated by the lack of effect with VEGF-infusion.

REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 2 OF 3 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 1998:275710 BIOSIS

DOCUMENT NUMBER: PREV199800275710

TITLE: Intramyocardial infusion of **FGF-1** mimics
ischemic preconditioning in pig myocardium.

AUTHOR(S): Htun, Patrik; Ito, Wulf D.; Hoefer, Imo E.; Schaper, Jutta;
Schaper, Wolfgang [Reprint author]

CORPORATE SOURCE: Max-Planck-Inst. Physiological and Clinical Res., W.G.
Kerckhoff-Inst., Dep. Experimental Cardiol., Benekestrasse
2, D-61231 Bad Nauheim, Germany

SOURCE: Journal of Molecular and Cellular Cardiology, (April, 1998)
Vol. 30, No. 4, pp. 867-877. print.

CODEN: JMCDAJ. ISSN: 0022-2828.

DOCUMENT TYPE: Article

LANGUAGE: English

ENTRY DATE: Entered STN: 24 Jun 1998

Last Updated on STN: 24 Jun 1998

AB Previous studies on the mRNA and protein level suggested a cardioprotective role of **FGF-1**. These presumed actions of **FGF-1** and **FGF-2**, as well as the underlying mechanisms, were investigated in this study. Human recombinant **FGF-1** (0.5 mug/ml, 20mul/min) and **FGF-2** (2mug/ml) were applied by means of direct intramyocardial infusion (IM) for 60 min prior to a 60 min LAD-occlusion and 120 min reperfusion. Myocardial infarction compared to the region at risk was significantly decreased by **FGF-1** and **FGF-2** treatment (**FGF-1**: 51.8 \pm 7.7%, respectively, **FGF-2**: 57.3 \pm 6.5% v control 83.4 \pm 2.8%, $P < 0.05$). The increase in survival time was about 33 min, and equalled that of **ischemic** preconditioning. This effect was caused by the mitogenic part of the molecule, since infusion of a truncated version of **FGF-1** (0.5-1 mug/ml), lacking mitogenicity but maintaining hemodynamic activity, did not induce cardioprotection (78.3 \pm 0.73% v control 83.4 \pm 2.8%). Suramin (0.5mug/ml) prevented the observed cardioprotection (77.0 \pm 1.2% v control 83.4 \pm 2.8%) proving that the cardioprotective effect is receptor-mediated. Genistein (0.5 mug/ml), an inhibitor of tyrosine kinases, abolished the cardioprotection as well (77.2 \pm 2.4% v control: 83.4 \pm 2.8%). Immunohistochemical staining revealed an uptake and translocation of exogenous **FGF-1** to a (peri-)nuclear localization in myocytes and into non-myocytes for **FGF-2**. We conclude that both **FGF-1** and **FGF-2** are cardioprotective (**FGF-1** being more active on a molar basis), and mimic **ischemic** preconditioning. Their actions are receptor-mediated and receptor activation is involved. Uptake and transport to a (peri-) nuclear localization, seems to be a pathway of minor relevance, since it could not be blocked by tyrosine kinase receptor inhibition. Tyrosine kinase-coupled receptor occupation in general is not protective as demonstrated by the lack of effect with VEGF-infusion.

ACCESSION NUMBER: 1999:645432 CAPLUS

DOCUMENT NUMBER: 132:31013

TITLE: Angiogenic therapy of acute myocardial infarction by intramyocardial injection of basic fibroblast growth factor

AUTHOR(S): Nagamine, Hiroshi

CORPORATE SOURCE: Department of Surgery (I), School of Medicine, Kanazawa University, Kanazawa, 920-8640, Japan

SOURCE: Kanazawa Daigaku Juzen Igakkai Zasshi (1999), 108(3), 336-348

CODEN: JUZIAG; ISSN: 0022-7226

PUBLISHER: Juzen Igakkai

DOCUMENT TYPE: Journal

LANGUAGE: Japanese

AB Angiogenic growth factors play an important role in the development of coronary collateral circulation. Therapeutic angiogenesis using exogenous growth factors may be useful in patients with severe coronary artery disease in whom complete revascularization is difficult. The present study was performed to examine the effect of one-time intramyocardial injection of basic fibroblast growth factor (bFGF) on myocardial blood flow, vascular d., and ventricular morphol. and function in a canine model of acute myocardial infarction. In 15 anesthetized dogs, myocardial infarction was induced by ligation of the left anterior descending coronary artery (LAD) distal to its first diagonal branch. In 6 of the dogs, **human recombinant bFGF** 100 µg was injected at 8 sites of the **ischemic** area of the left ventricular wall between the LAD and left circumflex coronary artery, while the 9 other dogs served as controls. Myocardial blood flow was determined using the colored microsphere technique both before and immediately after coronary ligation and again 3, 7, 14, and 28 days after treatment. Myocardial blood flow was expressed as a percentages of the normal flow. Cardiac function was evaluated by repeated echocardiog. measurement. Morphometric anal. was performed in excised hearts by assessing the infarct expansion index and ratio of thinning of infarcted to non-infarcted areas. Angiogenesis was assessed by immunohistochem. stain with anti-von Willebrand factor antibodies. Treatment with bFGF increased endocardial collateral flow in the border zone and the disparity between bFGF-treated and control dogs was significant on day 3 (74.5% and 40.8% in the bFGF and control groups, resp.), with a significant difference on day 7 as well (71.5% and 38.8% in the bFGF and control groups, resp.). Treatment with bFGF increased the epicardial collateral flow in the infarcted zone and the disparity between bFGF-treated and control dogs was significant on day 7 (67.4% and 35.7% in the bFGF and control groups, resp.). Histopathol. examination of the hearts 4 wk after ligation revealed that treatment with bFGF significantly increased the number of capillaries (39.7 and 22.7 per 200 + field in the bFGF and control groups, resp.) and arterioles (4.50 and 2.33 per 200 + field in the bFGF and control groups, resp.) in the border zone. It improved the left ventricular ejection fraction on day 7 after infarction (53.5% and 37.3% in the bFGF and control groups, resp.) and reduced the thinning ratio (43.5% and 26.4% in the bFGF and control groups, resp.). One-time intramyocardial administration of bFGF enhanced collateral vascular development, significantly increased myocardial blood flow and consequently improved global ventricular function. These results suggest that this method has potential as a new therapeutic approach in the treatment of myocardial infarctions.

ACCESSION NUMBER: 1998:24874 CAPLUS
 DOCUMENT NUMBER: 128:106303
 TITLE: Adenovirus-mediated expression of the secreted form of
 basic fibroblast growth factor (**FGF**-2)
 induces cellular proliferation and angiogenesis in
 vivo
 AUTHOR(S): Ueno, Hikaru; Li, Jian-Jun; Masuda, Satoko; Qi, Zhe;
 Yamamoto, Hiroaki; Takeshita, Akira
 CORPORATE SOURCE: Molecular Cardiology Unit, Department of Cardiology,
 Kyushu University School of Medicine, Fukuoka, 812-82,
 Japan
 SOURCE: Arteriosclerosis, Thrombosis, and Vascular Biology
 (1997), 17(11), 2453-2460
 CODEN: ATVBFA; ISSN: 1079-5642
 PUBLISHER: American Heart Association
 DOCUMENT TYPE: Journal
 LANGUAGE: English

AB Blood supply through collateral arteries is of critical importance in
 occlusive arterial diseases such as **coronary** atherosclerosis.
 Induction of angiogenic growth factor within either the narrowing arteries
 or jeopardized myocardium may promote angiogenesis in vivo, leading to
 salvage of **ischemic** myocardium. We constructed a
 replication-defective adenovirus (AdCasFGF-2) coding for **human**
 basic fibroblast growth factor (**FGF**)-2 that is modified, so that
 its secretion will be facilitated, by tagging a signal sequence derived
 from **FGF**-4. A large quantity of **FGF**-2 was
 detected in both the cell lysate and culture medium of COS cells infected
 with AdCasFGF-2; indicating that **FGF**-2 was secreted at least
 partly from the infected cells. The conditioned medium from the infected
 COS cells stimulated DNA synthesis in and induced cellular proliferation
 of arterial smooth muscle cells. These effects were eliminated by
 adenovirus-mediated overexpression of a dominant-neg. truncated
FGF-receptor type 1. Implantation of a gel of basement membrane
 proteins containing fibroblasts infected with AdCasFGF-2 into the ventral s.c.
 space of mice induced extensive cellular proliferation and the formation
 of functional arterioles. Cells surrounding the vessels were pos.
 immunostained with antibodies recognizing either smooth muscle-specific
 α -actin or factor VIII antigen as a marker for endothelium. These
 results suggest that AdCasFGF-2 may be useful for delivering functional
FGF-2 into tissues and may lead to therapeutic angiogenesis in
 vivo.

ACCESSION NUMBER: 2003:664593 CAPLUS
 DOCUMENT NUMBER: 140:139562
 TITLE: Gene therapy for **coronary** artery disease:
 preclinical and initial clinical results with
 intracoronary administration of Ad5FGF-4
 AUTHOR(S): Watkins, M. W.; Rubanyi, G. M.
 CORPORATE SOURCE: Cardiology Unit, University of Vermont College of
 Medicine, Burlington, VT, 05401, USA
 SOURCE: Ernst Schering Research Foundation Workshop (2003),
 43(Human Gene Therapy: Current Opportunities and
 Future Trends), 61-78
 CODEN: ESRWEL; ISSN: 0947-6075
 PUBLISHER: Springer-Verlag
 DOCUMENT TYPE: Journal; General Review
 LANGUAGE: English

AB A review which discusses the preclin. animal data and initial clin. in the
 largest population of patients to date who have undergone intracoronary
 administration of gene therapy for myocardial ischemia, specifically with
 replication-deficient adenovirus containing a **human** fibroblast
 growth factor gene.

REFERENCE COUNT: 16 THERE ARE 16 CITED REFERENCES AVAILABLE FOR THIS
 RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

ACCESSION NUMBER: 2003:610064 CAPLUS
 DOCUMENT NUMBER: 139:160389
 TITLE: Techniques and compositions for treating
 cardiovascular disease by in vivo gene delivery of
 angiogenic peptides and proteins
 INVENTOR(S): Hammond, H. Kirk; Dillmann, Wolfgang; Giordano, Frank
 J.
 PATENT ASSIGNEE(S): USA
 SOURCE: U.S. Pat. Appl. Publ., 51 pp., Cont.-in-part of U.S.
 Ser. No. 609,080, abandoned.
 CODEN: USXXCO
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 12
 PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------|----------|-----------------|----------|
| US 2003148968 | A1 | 20030807 | US 2001-847936 | 20010503 |
| US 5792453 | A | 19980811 | US 1995-485472 | 19950607 |
| US 6100242 | A | 20000808 | US 1997-722271 | 19971229 |
| US 6174871 | B1 | 20010116 | US 1998-132167 | 19980810 |
| WO 9940945 | A2 | 19990819 | WO 1999-US2702 | 19990209 |
| WO 9940945 | A3 | 19990930 | | |
| W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM | | | | |
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| AU 9947541 | A1 | 19991125 | AU 1999-47541 | 19990910 |
| WO 2001034208 | A1 | 20010517 | WO 2000-US30345 | 20001103 |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM | | | | |
| RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, | | | | |

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| | DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, | |
| | BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG | |
| ZA 2002003303 | A 20030526 | ZA 2002-3303 20020425 |
| WO 2002089856 | A1 20021114 | WO 2002-US13990 20020503 |
| WO 2002089856 | C1 20040401 | |
| W: | AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, | |
| | CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, | |
| | GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, | |
| | LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, | |
| | PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, | |
| | UA, UG, US, UZ, VN, YU, ZA, ZM, ZW | |
| RW: | GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, | |
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| | GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, | |
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| US 2004132190 | A1 20040708 | US 2003-741907 20031219 |

PRIORITY APPLN. INFO.:

| | |
|-----------------|-------------|
| US 1995-396207 | B2 19950228 |
| US 1995-485472 | A2 19950607 |
| US 1997-852779 | B1 19970506 |
| US 1997-722271 | A2 19971229 |
| US 1998-21773 | B2 19980211 |
| US 1998-68102 | B2 19980430 |
| US 1998-132167 | A1 19980810 |
| WO 1999-US2702 | A2 19990209 |
| US 1999-435156 | B2 19991105 |
| US 2000-609080 | B2 20000630 |
| WO 2000-US30345 | A2 20001103 |
| US 1995-481122 | B2 19950607 |
| AU 1996-50287 | A3 19960227 |
| WO 1996-US2631 | W 19960227 |
| US 1996-660387 | B1 19960607 |
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AB Methods are provided for treating patients with cardiovascular disease, including heart disease and peripheral vascular disease. The preferred methods of the present invention involve in vivo delivery of genes, encoding angiogenic proteins or peptides, to the myocardium or to peripheral **ischemic** tissue, by introduction of a vector containing the gene into a blood vessel supplying the heart or into a peripheral **ischemic** tissue. A kit comprising a gene therapy composition, a device for introducing the composition into a blood vessel or tissue in vivo, and a vasoactive agent is also claimed.